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Incidental Focal Acantholytic Dyskeratosis in the Setting of Rosacea

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Dear Editor:

Focal acantholytic dyskeratosis (FAD) was first described by Ackerman[1] in 1972 as a distinct histopathological pattern associated with various cutaneous conditions, and with classic histopathological findings including suprabasal clefting, hyperkeratosis and parakeratosis, and the presence of acantholytic and dyskeratotic cells at the epidermis. While FAD can be observed in many various cutaneous lesions including benign and/or malignant epithelial lesions, fibrohistiocytic lesions, inflammatory lesions, melanocytic and/or follicular lesions[2-4]. These histopathological findings may also extend into the surrounding tissues, which often appear to be clinically normal. A 42-year-old woman was presented to our department with multiple erythematous pruritic papules and tiny vesicles on her face. The lesions had been present for several years and aggravated 7 days ago. Physical examinations revealed multiple 2 to 3 mm, slightly spongiotic-appearing papules and tiny vesicles with serous crusts on the face (Fig. 1). Laboratory tests obtained at that time were within normal limits. A skin biopsy of an erythematous papule on the nose was also performed, and histopathological results revealed focal suprabasilar clefting and acantholytic keratinocytes in the epidermis, dense inflammatory infiltrates and vascular dilatation with solar elastosis in the dermis, and a negative direct immunofluorescence (Fig. 2). In the serial sections, we observe the same findings. Differential diagnosis for the possibility of polymorphous light eruption, systemic lupus erythematosus, contact dermatitis, and dermatitis artefacta should be considered. But given these clinical and histopathological features, a diagnosis of rosacea with FAD was reached. The patient was then admitted to hospital for treatment with doxycycline 100 mg and antihistamines. After one week, the lesions had remarkably improved. The patient was then discharged, and continued on the same therapeutic regimen for an additional month, bythe time all lesions were nearly resolved.

To date, the etiology of FAD has been attributed to numerous sources including hormones, viral infection, various immunologic factors, tobacco use, physical stimuli although the exact causative mechanism of this finding remains unknown. Other researches have suggested that sunlight and/or ultraviolet radiation may lead to the development of FAD[5].

In our case, we propose that the acantholytic dyskeratosis occurred secondarily to ultraviolet radiation exposure, given the lesion's location on the nose, a chronically sun-exposed area. Furthermore, chronic physical irritations may also have influenced, as the patient complained of severe pruritus limited to the affected area, the resulting excoriations which possibly lead to acantholytic dyskeratosis.

To the best of our knowledge, there have not been any prior reports of FAD associated with rosacea. We also contended that UV exposures combined with consistent physical irritation (i.e. excoriation) which represent two prime etiological factors contributing to the development of FAD in our patient. Therefore we report herein a case of FAD associated with rosacea and this report may provide additional explanation of pathomechanism in incidental FAD in the setting of rosacea.

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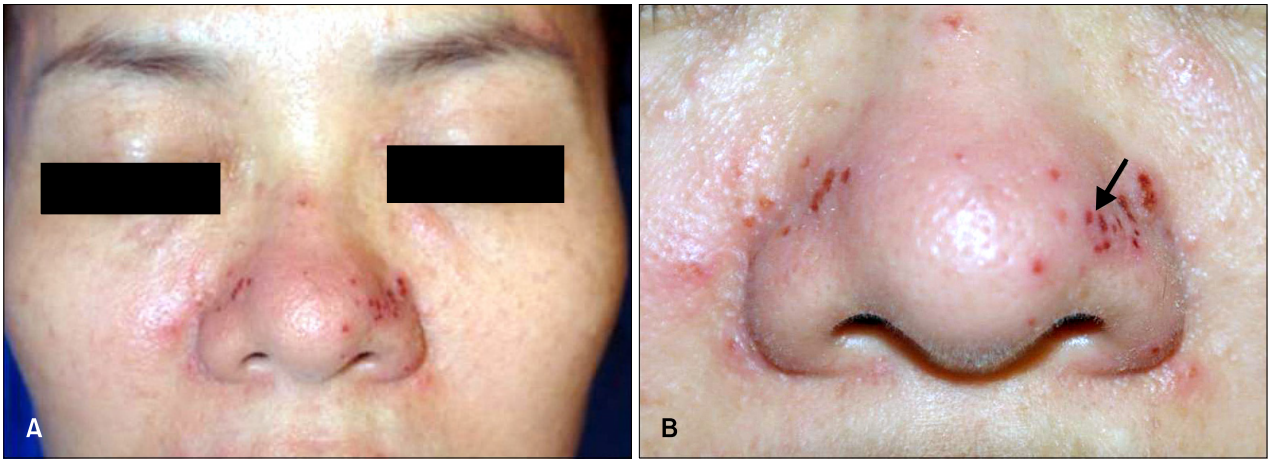

Fig. 1. (A) Multiple erythematous papules and tiny vesicles on the face, especially on the nose area. (B) Nose with diffuse erythema, multiple erythematous papules and tiny edematous vesicles. A biopsy was performed at the site of the erythematous papule indicated by the arrow.

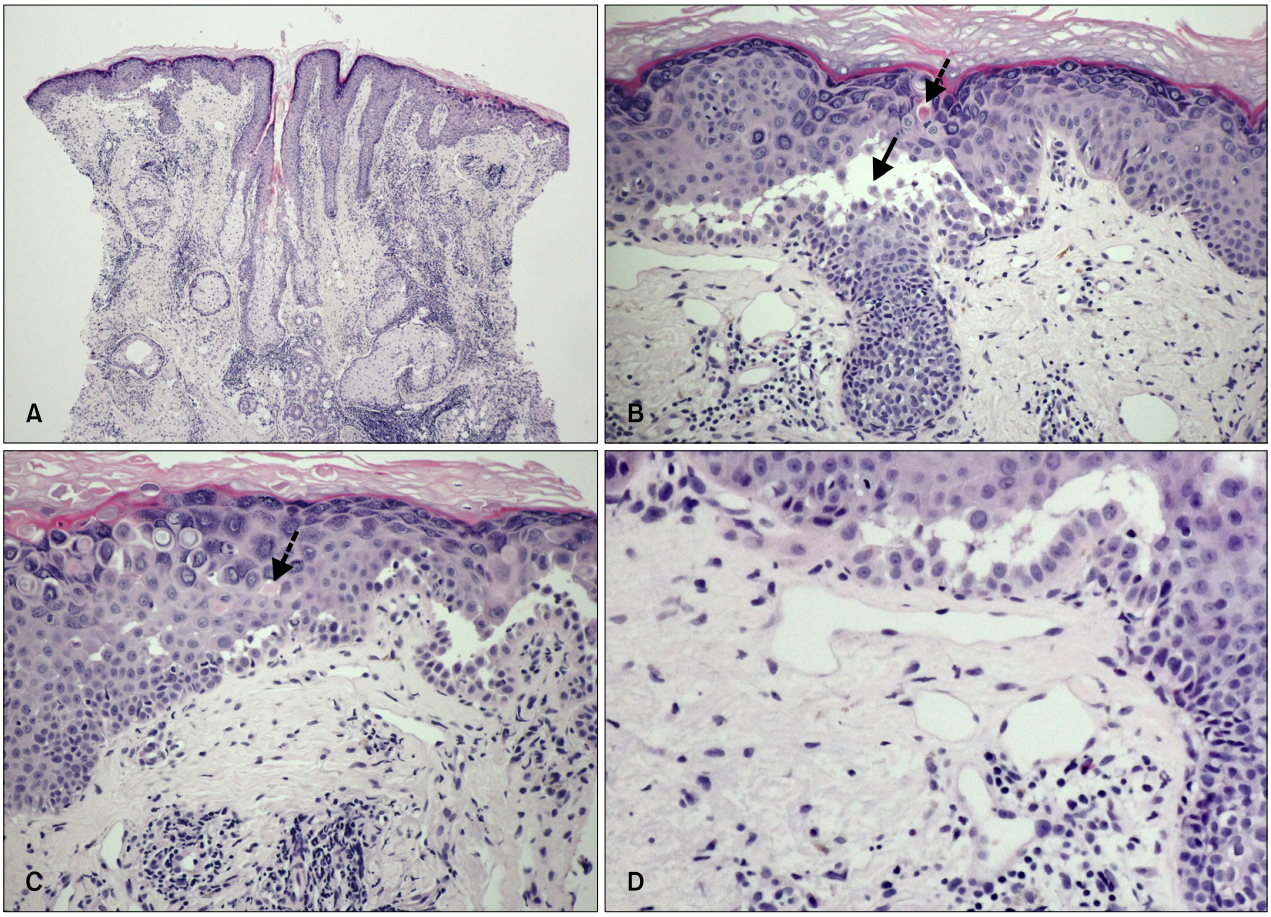

Fig. 2. Hyperkeratosis, acantholysis with suprabasilar clefting, with dermal vascular dilatation and inflammatory infiltrates (A; H&E stain, ×40). Epidermal hyperkeratosis and suprabasilar clefting, with scattered acantholytic (full lined arrow) and dyskeratotic cells (dotted arrows) occurring in the clefted epidermis (B, C; H&E stain, ×200). Dermal vascular dilation and inflammatory infiltrates with solar elastosis (D; H&E stain, ×400).

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Recurrent Milia-Like Idiopathic Calcinosis Cutis on the Upper Eyelid

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Dear Editor:

Milia-like idiopathic calcinosis cutis (MICC) is a distinctive type of idiopathic calcinosis cutis, and shows remarkable clinical and histological features. Most cases of MICC appear in children with Down syndrome, but cases of MICC unassociated with Down syndrome are occasionally reported[1]. Herein, we report a rare case of recurrent MICC after complete removal in a patient who had no evidence of Down syndrome.

A 17-year-old healthy Korean boy presented with a solitary whitish papule on the right upper eyelid for several months. Six years ago, he had complete removal of this lesion but the MICC recurred in the same area (Fig. 1). At the time of the patient's arrival at the clinic, physical examination revealed a 5 mm sized firm white papule (Fig. 1B), and it was noted to be similar to the milia that had been there before. His physical and mental development was normal, and he denied any history of previous trauma or dermatosis at the site of the lesion. Also, there were no specific findings in the past history or family history. Histologic examination of the biopsied lesion showed a condensed deposit of basophilic amorphous material within the upper dermis (Fig. 2B). Von Kossa staining showed a black colored reaction (confirmed as calcium) of the lesion, and the serial sectioning did not show the presence of an epidermal cyst. Laboratory findings, including the complete blood count, serum calcium, phosphate, and parathyroid hormone levels, were within normal limits, ruling out the diagnosis of metastatic calcinosis. With all the above findings, we diagnosed the lesion as MICC. After it was removed completely, there has been no recurrence for several months.

MICC appears as smooth, firm, whitish papules resem-

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